Etiology of Leprosy

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Original Communications.

I.—On the Etiology of Leprosy. By G. Armauer Hansen, Assistant Physician to the Leper Hospitals at Bergen, Norway.

The researches on which I shall here report are made with special respect to the occurrence of leprosy in Norway and to the opinions of Norwegian inquirers. As to these opinions being naturally just the same as those maintained elsewhere, this will hardly interfere with the general bearing of my arguments. These are discussed at full length in my report to the Medical Society at Kristiania; I cannot here enter so largely on details, and so my assertions may sometimes seem to be too little supported, but what I consider as essential facts will be brought forth with sufficient details for the reader to form his own judgment.

Leprosy is considered by different Norwegian writers as—

1. Not specific and hereditary (Danielssen, Boeck, Hoegh, Conradi, Bidenkap, &c.).
2. Not specific and not hereditary (Hjort).
3. Specific, miasmatic, and not hereditary (Hohnsen).
4. Specific, contagious, and hereditary (Lochmann).

What appears most striking in this discrepancy of opinions is that heredity is admitted as most essential as well by advocates for the non-specific nature of leprosy as by advocates for its specificity; and on the other hand, that one who considers it specific and another who considers it non-specific, do both deny its heredity. None of the writers on the subject, and this applies also to foreign ones) make any distinction between transmission to offspring of a specific and of a non-specific disease. And yet there is, in this respect, an essential difference in the phenomena, if we take as specific those diseases which are usually considered to depend on the operation of a distinct poison on the organism, whether this poison be a chemical one or a low form of organic life, including thus all parasitic diseases under this category. Of these specific diseases we distinguish acute and chronic ones, contagious and not contagious; all may be named infectious.

In direct contrast to the infectious diseases there is a long series of abnormal states which arise independently of any special influence, but which rather depend on a production of the organism not,

or only occasionally, influenced by external circumstances, such as many abnormities of skin, eyes, &c., many neuroses and mental diseases. It is satisfactorily demonstrated that all these states may be, and very commonly are, inherited. The phenomena connected with the hereditary transmission of these states are completely analogous to the phenomena by inheritance of physiological qualities.[1] In no case we consider the inheritance as depending on the transmission of any specific matter producing the same consequences in offspring as in parents, but as transmission of structural peculiarities. We use even the heredity of normal and abnormal mental peculiarities as a very weighty argument against the belief in anything specific-psychical, whereby every one at his birth, or perhaps before, becomes, so to say, infected.

If we now turn to the specific diseases, and examine how the case stands in respect of their transmission to the offspring, we need say little with respect to the unquestionably parasitic disorders, such as scabies, favus, &c., or of the acute infectious diseases. Although in many cases it would be a tolerably easy task to construct family tables exhibiting individuals in two or more generations who have suffered from scabies, ague, measles, &c., still no one has ever supposed heredity to exist in these diseases; they are in each case considered to be produced by the influence of the specific morbific matter. Variola can be transmitted from the mother to the unborn child, and so can, perhaps, some of the other acute infectious diseases, but this is regarded as contagion, no one thinks it to be inheritance.

Syphilis may be considered as a type of specific and contagious chronic diseases, and syphilis is generally considered the most exquisite example of an hereditary disease. And though the phenomena attending its transmission to offspring are, at least in most cases, just the same as those connected with variola, so the specific contagious disease is transmitted to the ovum. When it is sometimes assumed that congenital syphilis occasionally does not appear until some years after birth, nay, even till early youth, it is not, as far as I have been able to gather from literature, made apparent that the individuals in question have not had visible symptoms of the disease at their birth or shortly after it, and still less that they have had no syphilitic affections of internal organs. And to prove this must be difficult, if not impossible. It is a well-known fact that in autopsy often reveals to us a far-developed syphilis in internal organs, while no symptoms have as yet appeared outwardly in children of syphilitic parents. It would carry us too far out of the way, to enter upon

[1] Lucas, 'L'Hérédité naturelle.' Sedgwick, "Sexual Limitation in Heredity," 'Med.-Chir. Review,' 27, 28, 31, 32. Darwin, 'The Variation of Animals and Plants under Domestication.'

further details. I shall therefore only place side by side the chief phenomena attending the transmission to offspring of *Hereditary states* and of *Syphilis*.

1. The transmission is very often atavistic.	1. Atavism is not known.
2. Hereditary states manifest themselves at birth or in after years, and in the latter case mostly in corresponding ages in parents and descendants.	2. Congenital syphilis appears outwardly, when the child has not already died in utero, at or shortly after birth. At all events it does not appear to be established beyond doubt that no affections, internal or external, appear until in later life. There is no question of corresponding ages.
3. Hereditary states are often completely, or to a great extent, limited to the same sex.	3. There is no sexual limitation.
4. There is correspondence between the parts affected in parents and children.	4. There is no such correspondence.

These striking diversities must make us consider whether there be a real difference between the transmission to offspring of the acute specific contagious disease variola, and that of the chronic specific contagious disease syphilis. Upon a closer examination of known facts and upon grounds theoretical it will be found, I believe, that the incongruities are easily accounted for if the transmission is by inheritance in the one instance and by contagion in the other, and so in the latter in conformity to the transmission of variola.

One has only for a moment to take it for granted that syphilis is generated by a chemical poison, or rather by a parasitic organism, to admit the difficulty of forming a clear idea of a parasite being inherited.

Ergotism and pellagra are chronic specific non-contagious diseases. Pellagra is thought to be hereditary, not so ergotism. As to the etiology and the alleged heredity of pellagra, I beg leave to refer to Hirsch, 'Handbuch der historisch-geographischen Pathologie,' i, p. 478, &c., from which it must appear evident that there does not really exist any heredity. Further, pellagra is not transmitted to the ovum by contagion, a circumstance quite in harmony with the fact of its not being contagious at all.

The transmission to offspring of the diseases mentioned seems to me to stand in such decided relation to their etiology, as indicates a regularity which will most probably have universal application. My views on this head may be set forth as follows :

1. Those diseases which depend on a structural defect are hereditary.

2. Those diseases which are produced by a specific virus are of two classes, according to their character of being or not being contagious :

a. If the disease is contagious, it *may be* transmitted by contagion to the ovum, but is not hereditary.

b. If the disease is not contagious, it is not in any way transmitted to the descendants.

Though I cannot here go into particulars for evidence to support the above propositions, the reader will, perhaps, not find my conclusions too premature, when I conclude it highly probable that heredity and contagion are in direct contrast to each other. Engaged in investigations on the etiology of a disease the real cause of which is so entirely unknown as that of leprosy, the more circumstances I can bring forward to demonstrate the probability of one of the two alternatives existing or not, the more the probability of the other existing too will lose or gain. And this applies alike to other diseases. It ought to be remembered that Virchow always has maintained, specially for cancer and tuberculosis, that nothing specific is inherited but a disposition of certain tissues for the special disease. A predisposition to a special infectious disease can hardly exist; one man may catch syphilis, favus, &c., more readily than another, but that no one is born with a disposition for catching only one special infectious disease and no other, can hardly be disputed. On the other hand, contagion may be regarded as an unfailing test of a disease being produced by a special virus.

All attempts to point out the agents that in a specific or non-specific[1] way should directly generate leprosy, have hitherto failed. The discussion on the various alleged causes may, in my opinion, rightly be postponed till one of the two alternatives, heredity or contagion, be proved. I will mention, however, that all of them, including miasma and the eating of tainted fish, may, with a degree of certainty be shown to be insufficient to account for the occurrence of leprosy in Norway—may even be excluded. On the other hand, it can be shown that some of them, viz. uncleanliness, the occupation of the people, in short the whole mode of life of our peasantry, are very favorable to the spread of contagious diseases. For this we have well-established facts, such as the almost awfully common occurrence of scabies, though much on the decrease of late years; the spread of syphilis within families, in some instances most astonishing; the carrying about of typhoid fever by individuals among the fisher population congregated at certain times and places during the great fisheries.

It may also be mentioned that in the cases of leprosy in England and Germany represented as having been engendered in those countries, and of which I have read accounts and descriptions, the symptoms have not agreed with the symptoms of leprosy as they manifest themselves in Norway.

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[1] In Norway the assumed non-specific origin of leprosy is called spontaneous.

The assumption of heredity is based on the relatively frequent occurrence of several lepers in the same families. Among the 528 lepers registered by Bidenkap[1] there were 135 without leprous relations in the ascending line or in collateral lines, 268 had leprous relations in collateral lines, 125 in the line of direct ascent. This last proportion is nearly the same as that calculated by Dr. Hoegh for the whole country. Bidenkap says, "It seems thus to be something constant, applying to the whole country or to any single part of it, that from one fifth to one fourth part have had leprous ancestors, and at least about one fifth leprous parents." This seems to be confirmed by the proportion among the 210 cases now registered by me, among which 51 have leprous relations in the line of direct ascent, 50 no leprous relations, and 109 have them in the collateral line. My tables are taken from another part of the country than Bidenkap's, which seems still more to confirm the rule. The proportion, however, falls out very differently in several smaller districts, as it varies from one half to one twentieth, while in some places there are none with leprous ancestors; and it is, perhaps, remarkable that where the appearance of leprosy may be considered as relatively of recent date, there is very seldom any relationship between the lepers, nor do the latter descend from leprous families of any other places. It is not until leprosy has become endemic that the cases occur which might indicate heredity. If, now, the disease was contagious, this might have a very natural explanation, as the first case of leprosy acquired by contagion elsewhere would afterwards most frequently infect those in nearest intercourse, members of the same family or relatives. In any case the number of lepers for whom even with the strictest search no leprous ancestors can be discovered, and of those for whom no leprous relations can be pointed out, is so great that one fourth or one fifth of such as have leprous ancestors is too small a proportion to prove heredity. After what I have already stated as to the improbability of degeneration or special disposition to sickness in the people, or of any circumstance peculiar to us for contracting leprosy, it will by no means be unjustifiable to refer all cases without demonstrable leprosy in the line of direct ascent to those which cannot be classed in connection with heredity, and then the number of these cases will be so great that heredity will appear improbable.

With regard to pellagra, it has been endeavoured to prove heredity in the same manner by recording a great many family cases. But now it has been shown in respect of this disease that if individuals go beyond the influence of the home infection they do not only not get pellagra, but that even if they have previously suffered from it, the disease disappears under the altered conditions, while it

[1] 'Norsk Magazin for Lägevidenskab' (Norwegian Magazine for Medical Science), 2nd series, vol. xiv.

breaks out again if the party concerned be again exposed to the action of the specific infection. According to the numerous cases which, supposing heredity in leprosy, must be attributed in this disease to atavistic inheritance, the hereditary disposition must probably also be very slow to disappear; the same must be the case if we suppose a degeneration accumulated through many generations, or any other abnormity which might manifest itself without previous disease in the family. From whatever side we examine the supposed heredity in leprosy, it is therefore not to be expected that it should cease to operate, as it were, all at once, if the leprous family should be transported to another soil.

Prof. Boeck[1] has, as is well known, while acknowledging this, instituted his investigations among our countrymen in the United States, and is of opinion that the result of these investigations must establish the heredity of leprosy beyond doubt. The United States must be considered as certainly the best locality or such researches. It is sure that there has been no leprosy previously in those places where our countrymen have settled, and we cannot adduce local circumstances nor the conditions of life as causes of leprosy. What was to be sought for in America in order to prove the heredity, was cases that might be referred to atavistic inheritance from immigrants who themselves were free from the disease. If no such cases can be found then the heredity will not be made more probable than it was before, and in considering the possible cases of this kind it would be necessary also in America carefully to exclude contagion, for there are not so few lepers with the disease in mild forms who have emigrated to America.

The condition here suggested is not fulfilled in the cases reported by Prof. Boeck from America; in none of these can we exclude the possibility of the disease having been brought from home or acquired by intercourse with lepers in America (Observation 4). I must here again refer to the cases of leprosy mentioned by Danielssen and Boeck, and again quoted by Boeck in his last article, in which the disease broke out in a Frenchman and in a Dutchman respectively six and ten years after their arrival from leprous districts. This establishes undoubtedly that the disease may not come to light until many years after the time when it was contracted. All Boeck's patients were born in Norway, and the longest time after the arrival in America until the distinct manifestation of the leprosy was fourteen years. But in the face of the two cases quoted in which no heredity can be maintained, I cannot admit that fourteen years is a sufficiently long interval to exclude the possibility of the disease being brought from home. If one has only to depend on the patient's own statement as to the date of origin of the disease, then it is according to my

[1] 'Nord. Med. Archives,' iii, 1.

experience impossible to determine how great an interval may be allowed in this respect. We stand here before a great hiatus in our knowledge, which Prof. Boeck also admits, while he does not attribute so great importance to it as I do in opposition to the cases collected in America. But it is my conviction that also a great many of our lepers here at home are credited with too short a duration of disease. The patient's explanation is nearly always uncertain and hesitating. It happens rather often that anæsthetics indicate a very short duration of disease, as they reckon the commencement of the disease from the marked loss of sensation and the manifestation of atrophy. They have nothing to say about spots, and yet traces of the latter may often be observed with great ease; the patients have no idea that they have had large and permanent spots for a long time, for only such spots leave evident traces. Still more easily may this occur with slight transient eruptions, which, as a rule, have their seat on the back and on the extremities, especially with people who seldom or never wash themselves.

With tubercular patients it happens that they are conscious to having had slight eruptions or a slight discoloration in the face many years before the manifestation of the disease.

According to experience hitherto such slight but yet suspicious skin eruptions have most nearly the character of an erythema nodosum. I shall briefly quote some cases of the disease in order to illustrate this.

L. H—, No. 804. Mons. Randal, spotty leprosy. Had, about three years before the affection existing when he was admitted, an eruption of red spots, tender to the touch and disappearing after about three weeks; at the same time pains in his limbs. A similar eruption in the following year. Again in the following year in November a new eruption, and of the spots then produced there were at his admission January 1st, 1873, only slight traces, from which leprosy could not with certainty be diagnosticated. More certain signs of the disease were the swellings of the inguinal and axillary lymphatic glands, and a slight insensibility along the outside of the backs of both feet, of which the patient himself had no conception.

L. H—, No. 731. Aslak Ljom. Tuberculous leprosy. Admitted September 14th, 1869; dates his disease from the spring, 1868, when there appeared tubercula on his thighs.

In the spring of 1863 he had an eruption of red tender spots on the extremities, which lasted from four to five days; a similar but not so strong eruption in the autumn of the same year and in the spring of 1864.

Now, I consider that the leprosy in this last patient began in 1863, but he himself considers that it began five years later.

I have already, in 'Nord. Med. Archives,' communicated cases as proofs that many years after the smooth form may seem to

have come to a conclusion and the spots and likewise the insignificant anæsthesia have disappeared, or possibly the latter has remained stationary, a relapse may occur in the form of a tubercular eruption, and this may take place after the lapse of five, six, or perhaps more years. If, now, a patient has no knowledge of his spots nor of his anæsthesia, as was the case of the patient first mentioned, and as is very commonly the case even with much greater degrees of anæsthesia, or if he has had for a year or two slight skin eruptions which he has not much noticed, and as the relapse or the continuation may not take place until many years afterwards, I cannot consider ten to fourteen years as any security for the disease not having been taken from home to America. I do not, therefore, base the justification of my assumption in this respect on an unlimited period of incubation; on the contrary, there is reason, according to my other observations, for believing that this period is not so very long.

I suppose, therefore, that the Frenchman and the Dutchman, as well as Prof. Boeck's patients from America, have had symptoms of leprosy before the time indicated by them, without being aware of it. It must be remembered that it does not so seldom happen that patients with syphilis are in the same case. In the question of contagion this circumstance is also of great importance. Even if the apparent time of the duration of the disease in one case and its occurrence in another is far apart, the latter may still be attributed to infection from the former. Moreover, the cases of the Frenchman and the Dutchman are in my opinion sufficient of themselves to make Prof. Boeck's cases not conclusive for heredity.

We are here in this country not without localities that in a certain degree may be compared with America—I mean our towns in the west country—and we shall now see how the case stands in Bergen, where there are many who have moved in from leprous districts. The numbers are not so very small. Among the patients (about 250) at present in the Asylum No. 1 there are no fewer than 119 who have near relations dwelling in Bergen; namely, 318 brothers, sisters, uncles, aunts, and cousins. If the children of these are reckoned, the number may be safely taken at 500. It would next be important to know how many of the other inhabitants of Bergen, where the working population is mostly recruited from leprous districts, may be reckoned as belonging to leprous families, or only how great a part of the population comes from leprous districts. Neither of these points can be settled. But it is a certain fact that the immigration to Bergen from leprous districts is of old date, although it has much increased in later times, yet the number of immigrants must previously have been very great; we constantly find among the working classes people whose parents or grandparents were immigrants, and the very general custom of giving to children the father's first name, with son or daughter affixed as surname, bears testimony to this also. And when we

have now seen how many there are who have relations at present in the asylum, it is scarcely too much if we reckon those inhabitants of Bergen who belong to leprous races by thousands. Here are, therefore, rich materials for heredity.

As regards Bergen, no change of climate, and hardly any change in the conditions of life in other respects, can be adduced, as the large majority of immigrants belong to the working classes. A great number of these are quite as much exposed to injurious climatic influences as the population of the country districts, and sometimes even more. The dwellings are, to a great deal, quite as bad as in the country. The only thing which can be alleged as an advantage of removal is a relatively larger consumption of fresh meat and greater cleanliness. The gain is, especially in this last respect, considerable. The itch is, according to my experience of two years as a physician among 2000 to 3000 of the working classes, of very rare occurrence, while this disease is in the country districts still very frequent, although much less so during the last few years. According to the same experience the change of food seems, on the other hand, to have less influence, for chronic derangement of the digestive functions is so common a disorder among the working population that it can scarcely be more frequent among the country population. As to the general state of health, it is incomparably worse in Bergen than in the country districts. Scrofulous affections are very common, and mortality is much greater. The soil seems thus to be quite as favorable for the fostering of leprosy as in the country districts, unless cleanliness is the chief factor.

But leprosy occurs in Bergen; and, then, the question presents itself whether we can say that it arises spontaneously there. If that is the case, there would be so much the greater probability for heredity being also an attribute. There have been, since 1856, noted more than fifty cases of leprosy in Bergen; the preponderating number of these (about forty) are people who either have gone out from the asylums and have settled in the town, or such as have had the disease in a manifest form only a few years after their immigration. These cases have no significance for us. As to the others, on which I defer communicating details, some of them ought to be considered as undoubtedly spontaneous cases according to common view, and for not a single case of leprosy among natives of Bergen can heredity be pleaded with any certainty; and this is quite striking when the materials for inheritance are so abundant, and when the spontaneous unspecific occurrence would appear to take place; these two modes of origin ought to go hand in hand. And yet the family conditions are what our attention has been almost exclusively directed to. This does not serve to support the theory of heredity.

But neither spontaneous occurrence nor heredity have much to

rest on when we can also show other cases for which contagion furnishes the most probable explanation. All information is wanting as regards possible contact with other lepers; and for this there has been in Bergen, and still is, tolerably abundant opportunity, as the inmates of the various asylums have more or less limited intercourse with the town, while the inhabitants of the town have also access to the asylums.

In the last few years the following four cases of leprosy have been quoted in Bergen—

1. Anders Hegrenaas, 63 years old, labourer, tubercular form; born in Jólster; states the duration of the disease to have been about three years in 1871. Does not know any leper in the family, which is also probable, as leprosy is very rare in Jólster, and he is not related to any of the lepers who have been registered since 1856. He has been in Bergen more than twenty years; quitted thirteen years ago his service at Lungegaard Hospital, where he had been bathing attendant four years, and therefore had been in close contact with the patients.

2. Anne Larsdatter Starefos, 51 years old, tubercular form; came twenty years ago to Bergen, and dwelt with a sister at Starefos, a farm which lies high up on the Floifjeld, close to the Asylum No. 1 and the Lungegaard Hospital. She was washer-woman at the Asylum No. 1 for ten years, and three years after she ceased to be so her leprosy was undoubted, but she had, however, long been suspected of not being healthy. She earned her living at that time by washing bottles in a beer brewery.

She was born on the farm Munie, in a mountain valley about two miles from Bergen. All people there are agreed that there never has been any leprosy in the valley. It is in any case certain that her parents, their brothers and sisters and parents, have all been healthy, and that she is the only leper known in the families from which her parents descend.

3. Joachim Berentsen, sailor, 26 years, tubercular form. His parents, who are still living, were born in Bergen. The parents of his sixty-years-old father (Lars Andersen) were both born in Indre Holmedal; they moved so early to Bergen that all their nine children, of whom the patient's father is the youngest, were born in Bergen. There are dwelling in Bergen grandchildren and great-grandchildren of them, who are all healthy. The mother's parents were from Horningdal, where there is no leprosy, and from Bergen. They had two daughters, of whom one has a healthy daughter, and the other has, besides our patient, two healthy children living. The patient resided at the age of fifteen years, in 1858-59, for half a year at Spidsöen, and during that time had intercourse with the peasants round about, and also with families where leprosy had existed. Kari Spidsöen, a girl of about the same age as the patient,

had then many leprous spots, and she and the patient played constantly together. It is not supposed that any more intimate connection took place. After his return to Bergen the patient worked on the Tyskebrygge, and very soon after his return, about half a year, his father remarked in his son an altered complexion, which aroused his apprehension of leprosy. This apprehension became certainty when his son went to sea in 1863. The patient himself became apprehensive of leprosy at the end of 1864, after having suffered from ague in Sulina, but he did not discover any undoubted marks of the disease until in the course of the year 1865.

4. Lyder Eriksen, carman, 27 years old, tubercular form. He was born in Bergen, a natural son of healthy parents born in Bergen; has a healthy sister born of same parents. The mother's father was from Aunland, in Sogri, and the mother from Hallingdal; the latter was not aware of there being any leper in her own family. The father's mother was of German origin, but his father was from Voss; he was healthy, but I have not been able to obtain any other information as to his family. There are dwelling at present in Bergen not a few relations of these families, and they are all healthy excepting our patient.

The patient was during a part of his childhood taken care of at the farm Lone, about two miles from Bergen. There were no lepers here, but at the neighbouring farm Espeland there was one, and the patient was often in contact with him. The patient was badly taken care of at Lone, and suffered *inter alia* from favus and scab. About eleven years ago he came back from Lone, but was there again for a short time the year after. The patient states that the disease broke out in 1870, that is, about four years ago; but his old grandmother, about eight years ago, called his mother's attention "to a bluish discoloration over the eyes, like a shade," which she did not like, and was of opinion that the lad was not all right.

As, now, heredity in leprosy is said to manifest itself until the fourth generation, and perhaps longer, nay even that it can operate without leprous ancestors, it is, of course, impossible absolutely to exclude this hypothetical heredity in any of the cases here cited, as it is almost impossible to exclude it in any case whatever in a country where leprosy is endemic. For the three last-enumerated patients it is, however, certain that there are two previous healthy generations in the family, and extremely probable at least that the respective patients are the only lepers in the families. None of them can be said to have lived in specially unfavorable conditions of life, except, perhaps, No. 4. But I have succeeded for them all in showing contact with lepers; and as regards one of them (No. 3), that the first indistinct traces of leprosy manifested themselves a very short time after he had been in contact with a leper.

It must certainly be admitted that the most probable explanation of the origin of these cases of leprosy must be sought in infection at the respective places where the parties concerned have been in contact with lepers. There is no holding point for miasma nor for infection by food, especially in Cases 1 and 3, and there remains for us only that mode of infection which we usually designate as contagion. For No. 2 (the washerwoman) there is good reason to suppose that the contagion may be communicated by things which have been in intimate contact with lepers.

If we now consider the occurrence of leprosy in Bergen on the whole, most of the cases are such as may be considered to be imported from the country; then relatively few cases affecting the natives of the town, which cases many would call spontaneous; no case which with any certainty could be referred to heredity; and, finally, four cases, two of patients born in the country district and two natives of the town, which with the greatest probability may be attributed to contagion. It must be remembered that, assuming the heredity of leprosy, we might expect with such abundant materials to find a large number of lepers. It appears to me that what is here pointed out does not tell for heredity, but even tells very strongly against it. The cases which stand without any reasonable explanation I have for my part no hesitation in referring to contagion, and this seems according to the data before us much more justifiable than attributing them to inheritance, for which we have not been able to find a single point of support on the spot.

We shall now transport our investigation to some of the homes of leprosy in the country districts. The task will be still the same—to weigh probabilities against each other; it is still here more difficult to obtain any certain proofs, and I suppose the reader will now agree with me that it is far from sufficient to show relationship between lepers in order to establish by inference the hereditary origin of the disease.

As above mentioned our peasants' mode of life in general is very favorable for the transmission of contagious diseases, and while leprosy is so frequent it is just as impossible to exclude contact with lepers as to exclude heredity in the third or fourth generation. The difficulty arises chiefly from the circumstance that leprous patients may suffer from the disease for years without themselves or others knowing it. In order to illustrate what long intervals in accordance herewith must be allowed for considering a contagion possible, and which makes it so difficult to discover any special occasion of contagion where opportunity for contact with lepers is so abundant, I shall begin with a case from Volden, in Sondmore.

As the only lepers in a farm Solliden we find Peder Solliden, his wife, and his wife's second husband; the second wife of the latter is still living and is healthy, as also all the children of these marriages.

On examining the surviving wife, as well as other people who had known the parties concerned well, I could not find that in three generations there had been any other lepers. The first leper, Per Solliden, rowed during the fishery for many years together with Elling Staaten and Martin Roünestad, who were both lepers, and there was unanimous testimony to Martin having been a leper for many years when he and Per Solliden were constantly chumming together both winter and summer, while Elling Staaten probably became leprous at a later date. Per Solliden died a leper in 1841; his wife married again; when she became a leper is not known, but she died a leper in 1856, and it was not until 1860 that her second husband was aware of his own leprosy.

Solliden is a mountain farm, as the name denotes, situated on the sunny side, and the situation must thus be considered as favorable. As no leprous relations could be indicated for any of the three, it becomes probable that they have been infected by contagion. In this case there would be a series of contagions for three cases, from the close of the year 1830 to 1860, which at first glance seems improbable. But I suppose that the woman who died a leper in 1856 was already a leper when her husband died fifteen years previously, which is no improbability, whether she had the disease in the smooth or tubercular form, and that she was not aware of it when she married again. Between her death and her second husband's manifest leprosy there is only an interval of four years. And as analogous to this I can cite the following case:—Knud Villa, Tresfjorden, in whose family no leper can be found, rowed during the fishery with Ole Sætre and lay with him in the same bed when employed in building work in the summer of 1858. In 1859 the leprosy of Ole Sætre was so far advanced that he was sent to Reknæs Hospital, and in 1864 Knud was no longer in doubt as to his own leprosy; he is a strong man and lives in tolerably good circumstances. Also in this case contagion seems to be the most probable explanation.

While in the cases mentioned no leprous relations could be indicated, we have in the following three leprous children of the same leprous father, where also contagion may be made probable, although the presumption seems to be in favour of heredity. All these have been in contact with a leprous servant girl from Stryen, who served at the farms Rake, Aflem, Bruvold, and Algældet in Indre Nordfjord. At Algældet she lay in the same bed with the daughter of the house, Malene, and the latter became diseased at home in 1850 or thereabouts; after her the father became diseased in 1859, and at last two sons in 1866 and 1867; both sons have lain in bed with their father. The idea of attributing this to contagion from the servant-girl may be combated by the fact that the girl when serving at Bruvold, while she was undoubtedly leprous, attended to the youngest son in the house, who is still living and is healthy.

But the notion may be corroborated by the circumstance that one case at Rake, Anne, the only one in the family, can be brought into connection with her, as Anne during the summer, while at the mountain pasture, lay in the same hut with the servant-girl when the latter was at Rake, and in the same hut people lie also in the same bed. Anne became aware of her disease about eight years after she had lain at the mountain pasture with the servant-girl.

While I remark that, according to observations of near relations for two of the cases reported from Bergen, it must be considered certain that the disease exhibited slight symptoms already four or five years before it was noticed by the patient, we shall not henceforth require to dwell further on the very important objection which may be drawn from the apparently long intervals occurring between the particular cases which may be supposed to have communicated the infection to each other. I have stated what I could discover to refute the notion, and I will in the following lines only insist on those other points which may have some influence on the question between heredity and contagion.

I have met with two cases of immigrants into leprous districts from non-leprous districts who have got the disease, and who both can be proved to have been in contact with lepers. One of them is Oline; she was born in Sunelven, where leprosy does not occur, so that inheritance may with tolerable certainty be excluded. Since her residence in Sombrefjord she has had frequent intercourse with many lepers, especially with those at the neighbouring farm, Langsten. The other is Petter Jensen, from the parish of Straud, where also leprosy does not exist. He has constantly attended to, and taken care of his leprous neighbour and brother-in-law, Petter Riksheim; he tended him when dying and as a corpse, and became leprous shortly after.

For these two cases it seems, therefore, that contagion furnishes the most probable explanation; they had both immigrated to leprous regions, and we come thereby to that view of the case which Holmsen defends, namely, that leprosy is not attached to the families, but to the place. I shall in the following lines adduce several cases which tell for that notion; but as I must repudiate an explanation by help of miasma or of special conditions of life in these places, I seek the probable explanation in contagion.

The above-named Petter Riksheim belongs to a family[1] in which there are many lepers. The family comes from the estate Hjelle in Orskoug; and in this part of Orskoug no leprosy occurs, as also the old Lars Ellingsen Hjelle maintains that leprosy never has been known at Hjelle nor in his family. All the members of the rather

[1] I use the designation race or family to distinguish in some degree between the greater groups of relations. By family I usually mean the collection of families descending from one pair; by race many families grouped together.

extensive family who have become leprous, have now removed westward to regions where the disease is at home, while those who have remained near the family home are free from the disease.

Still more evidently does this appear in a large family at Volden. It is a family that comes from the before-mentioned farm Solliden; the family is so large that not nearly all its members have been noted; theleprous Rasmus was of opinion that there were about 150 cousins, and of these there are again many children. Now, in all this extensively ramified family, there is only leprosy in two houses; in one it is the mother, a son, and a daughter, who are leprous, and these dwelt at the end of a long fjord, where there were lepers before, partly on the same farm and partly on the neighbouring farm; in the other house there is only one leper; these last dwell in another village, but the leper has constantly rowed during the fishery with his leprous cousin of the house first mentioned. Leprosy seems thus to be less attached to family than to intercourse.

In Olden, in Nordfjord, there live two families, and in Stryen one, who by marriage have become connected. In the latter and in one of the Olden families there is a leper in each; for the one case there have been three and for the other two previous healthy generations. In that part of Stryen where the one leper is, leprosy is relatively of frequent occurrence. I have visited the two Olden families, and I have seldom seen so fine well-grown and powerful people. A daughter of one of these families is leprous; she served at the house of a priest in Daviken, together with a lad who was obliged to leave his place on account of his leprosy, and who died shortly after. According to the usual custom among servants, she attended this man, made his bed, washed and mended his clothes. Also in these cases I must consider hereditary disposition as not very probable.

At the farm Bjorlo at Nordfjordeidet, which, like all farms here, lies on a terrace and is thus very dry, there have been two brothers and the son of one of them lepers; moreover, a more distantly related woman who dwells in the neighbourhood is also leprous. Here the relationship seems to be of importance, and likewise for the two children in another family. But no leprous ancestors are known for any of them. How the first leper at Bjorlo could possibly have got the disease it is not known, neither is any information to be had with regard to the more distantly related woman, but the second at Bjorlo has, during the building work, lain in the same bed with his leprous brother, and the son has naturally had intercourse with his leprous father, with whom he lived; and of the brother and sister, the sister was married with a leper, and the brother is brother-in-law to the two leprous brothers at Bjorlo, where he also dwelt and had intercourse with them. All the cases at Bjorlo, as well as the leprosy of the female relation in the vicinity, fall in the interval from 1843 to 1856.

Also here it seems that the locality and contact have equal weights in the scale with the family conditions.

The farm Möklebust at Nordfjordeidet is situated, like Bjorlo, on a terrace close to the sea. At this farm there have been, in the earlier part of the century, some lepers; descendants of this family are still living, some of them at Möklebust, and they are healthy. In the later time there have occurred three other cases, one in a family very well off, the only case known, and two likewise, the only cases, in another but poor family. These cannot be brought into connection with the other lepers at Möklebust, but in 1845-46 two of the leprous girls from Hjelmelandsbakken were servants at the farm; the one leper at Möklebust was at that time a young child, and had intercourse with the two leprous girls from Hjelmeland; ten years afterwards her own leprosy became evident, and eight years later the leprosy of her brother and of the third, Brite, was also manifest, she having been in intercourse with both.

Here are at least three cases of leprosy without any reasonable holding point for heredity, but with undoubted contact with other lepers.

I have registered the lepers who for a long time have been and still are at Aalfoten. In a bend of the fjord lie the houses of Shoreim in a crescent on the east side, and here may be observed the rare case that all the inhabitants belong to the same family; opposite on the other side lie the houses of Ise, which are occupied by different families, and in Aalfoten properly so-called, which lies round a little bight of the fjord, are situated Vik, Sigdestad, and in the innermost part Möklebust. If we now take the leprous cases only according to family relationship, we have only, out of fifteen, three cases which are without leprous relations. But if regard be had to the conditions of locality and habitation, we shall find in Shoreim six lepers in one family, and elsewhere eight (nine) lepers in six different families. One of them has changed his residence; but it is not known whether he was leprous before moving or not. Where the whole soil is occupied by one family, there leprosy is attached to that family; but where the farms and the various cultivations of the farms are managed by branches of different families, there leprosy extends over six families, while the number of cases is only three more; and it cannot be because the families are not numerous enough; some of them are as large as the Shoreim family, so that there seems to be quite material enough for heredity. But with heredity as explanation we do not come far enough, and we come incomparably further with contagion. At Thoreim contagion can only affect one family, and there we find many cases of relationship, enough to form what we may call a thoroughly leprous family. In the other farms we find many families exposed to contagion, and no less than six are affected. There is no want of more

direct indication of contagion for one case: Lars is the only leper in a large family, but he lived many years in the same house with his leprous father-in-law.

That leprosy is thus attached to the locality and not to the family, is still better shown by the following instance. In the parish of Bredheim, in Gloppen, there are three farms, Skreppen, Skinbo, and Sætre, situated high up in the hill on the sunny side and close to each other. Considered as a hamlet, they may be compared with Thoreim, but the conditions of habitation are quite different, for they are occupied by members of many different families, as is usual; for the families dwelling in the same homestead seldom intermarry; they marry usually with those who live a good way off. From these farms there are found eight lepers in five different households not related to each other; to these may yet be added three not noted here, each belonging to a separate house, in all eleven lepers in eight different families. Even if there may be found in other places leprous relations of these families, the circumstance would still be remarkable, but according to report there are no such leprous relations, and as regards two of the families, I have been able to verify the accuracy of the statement. If the conditions of habitation had been as at Thoreim, we should certainly in these farms have had fine family tables.

I can further name two similar cases, though not so strongly characterised. At the farm Reed, also in the parish of Bredheim, there has been leprosy in various families. In one there are not less than five cases. The family is large, and its branches are spread about in various places, but only at Reed is the family leprous. The oldest leper, John, lived with his brother David; the two daughters of the latter became leprous, and likewise a cousin born on another farm but reared in David's house, and finally another cousin who dwelt at Reed.

At Œstrem, in Gloppen, leprosy appears in five cases in three families. Two of the lepers have a leprous cousin on the mother's side; but as this cousin had a leprous uncle who dwelt in the same house, this relationship is not of much importance.

Considering that in a relatively very short time and in a rather limited territory I have been able to collect so many cases where the peculiar conditions of habitation and of the distribution of families seem to play an important part for the appreciation of the occurrence of leprosy, I must assume that this manner of criticising heredity must have its significance. It is here in the west country most usual that the several occupants on the same farm dwell together in one homestead, and it is just as usual that these occupants are not related to each other. The homesteads are usually remarkable for being extremely uncleanly, and their inhabitants are equally so. In the last few years a favorable change has begun to take place by repartition, as this generally involves removal from a

common homestead, and I have repeatedly had occasion to see that removal brings about a better domestic arrangement.

To trace the history of the occurrence of leprosy in the several districts in this country is nearly impossible. There are, however, some circumstances which indicate that the disease did not until quite lately extend to certain districts; this applies specially to Finmark, and here leprosy attacks, not only Norwegians, but also Ovans and Fins; here the lepers are not very frequently related. Now, it is only in the last decenniums that the traffic with these regions of the country has become more lively, and that the great fisheries in East Finmark have been frequented in a large scale by fishers from leprous districts of the country. This might indicate an importation of the disease. Also in Sondfjord there is a locality where leprosy does not seem to be of old standing, namely, Jölster; the number of lepers is small, and only few of them are related; most of the lepers here have served or rowed during the fishery in and from the strongly leprous districts near the sea. Nay, in one of these latter, namely, Nöstdal, it is maintained that leprosy is not of older date than from about 1820. I received in that respect completely similar accounts from several old people in the various parts of the valley. The first leper in the valley is said to have been a woman from the neighbouring village, Solliden, who moved into the farm Indre-Koame; the second was a woman from the neighbouring farm Koame, who moved into the upper part of the valley; the next two cases occurred again on the farm Indre-Koame. If we could depend on this account it would indicate an importation; the people who gave the information were more than seventy years old, and the tradition was similar in the upper part of the valley, in the middle, and in the lower part down near the sea. Now, Nöstdal is, or was a short time ago, one of the most leprous districts; there are only a few farms in the thickly populated valley that have escaped the disease. The habitation is the same as usual; the houses are collected for the most part in homesteads; those who dwell in the middle of the valley intermarry mostly with those who dwell in the higher or lower parts, and *vice versâ;* this produces in the course of time such a combination of families that in the short time I could dispose of, it was impossible for me to attempt to get a clear account of them, as the valley contains between 3000 and 4000 inhabitants. Nevertheless, it appears, according to the information we possess, that the number of lepers without any leprous relatives is not so little (about one fifth). If, now, leprosy is contagious, then both the circumstance that there may be found strongly leprous families, and the circumstance that there may be found other families in which only a few or single members are leprous, will find a natural explanation. For if leprosy comes, as here, into nearly every homestead in the valley, it may in different homesteads affect the same

families, and in the same homesteads affect several different families, just as we have seen in the farms Skiuls, Skreppe, and Sætre, in Nordfjord. When the families are very much intermingled, it can excite no wonder that we have more family cases than isolated cases. With heredity as explanation these latter cases become quite enigmatical, while on the hypothesis of contagion the family cases will, on the contrary, not only not be surprising, but will be very easily intelligible. I shall, in further demonstration of this, now describe what may be observed in Tresfjord, in Romsdal.

I have there found three powerful races issuing from the three farms Skjærsvold, Eidhammer, and Bradstad, and the members of which are found spread round about the fjord and partly beyond. I have noted down more than 300 members, but several branches are not specified because no leprosy is found in them. The tables extend a good way into the former century; great-great-grand children of the eldest noted are now mostly middle-aged, and some of them old persons. A man of the eldest generation was married for the second time with his first wife's sister's daughter, and from this marriage there are now great-great-grandchildren, who will soon be marriageable. If we reckon in the members of these races not registered, we may probably calculate between 600 and 700, among whom there have occurred fourteen cases of leprosy. I do not think any case has escaped my notice, as the old woman (above 70) from whom I have most of the names indicated, had about the 300 names in her memory, and the accuracy of her memory I have been able to confirm by inquiries elsewhere as regards a great part of the information given. In spite of the extent of the races there are relatively very few interior marriages, and there are thus a great many importations by marriage; only for one family I have been able to find leprosy which might be brought into connection with two such importations, but that this connection can have any influence on the bringing in of any hereditary disposition is very doubtful, as these lepers, who were outside of the great races, all except one dwelled on the farm Vike, where four of the fourteen cases of leprosy had occurred within the races, and in four different families, of which three are related nearly, and the fourth more distantly. If we now follow the subject on which we have entered, namely, that of local circumstances, we shall find, besides the four in one farm, three more children of common parents in one farm, two in another, and two nearly related in two neighbouring farms; these two have had intercourse together, and in the house of one of them a leprous man has been servant and had his bed and clothes attended to by the party concerned.

There remain thus only three isolated cases; two of these are of ancient date, about 1830; on one farm, where the third case occurred, there has been leprosy before, and there is still one leper.

Leprosy occurs thus neither much diffused in the many families of these races, nor in many places in proportion to the diffusion of the races. The proportion is not very different from the proportion between the lepers and the inhabitants, otherwise this cannot be determined accurately, as it is not possible to obtain such accurate information about lepers from former times. Moreover, the same tradition exists in Tresfjord as in Nöstdal, that it is not more than fifty or sixty years since leprosy came into the fjord.

We have for these races as good probability as in general can be obtained for there not having been leprous in previous generations, and we find nowhere parents and children simultaneously lepers. In regard to the very irregular heredity, and to the presumption necessary for heredity of there having been leprous ancestors in the previous century, since neither with respect to these races nor indeed to any other can the slightest probability of progressive degeneration or anything like it be shown by any one, this occurrence of leprosy appears susceptible of natural explanation by help of contagion. While these three races are spread everywhere round the fjord, no contagious disease which affects a greater number of the farms situated here can well avoid affecting members of these races. The contrary would, indeed, be remarkable. Such an interpretation is also supported by the appearance of cases in groups in races which can by no means be designated as thoroughly leprous; indeed, such races can scarcely be said to exist; very leprous races are extremely rare, while, on the contrary, the more limited family may be so, and then it is most usual that several brothers and sisters, chiefly those of more nearly the same age, are the lepers. Of this there are three instances in these races, and I have before shown by instances that such may find explanation by help of the habitation of the family and contagion, while the other families of the same line may be quite exempt. The circumstances in Tresfjord do not otherwise offer anything particularly noteworthy; the lepers of whom I have been able to get an account, are partly the only ones of the family, partly there are only a few in the same family; the same case as elsewhere, where the families are not large or leprosy not very prevalent.

I could yet make commentaries on some of the cases collected, but partly these would be incomplete and partly they could not give us any new points of view. I will only remark that I cannot point out any case in which heredity has any preponderating probability for it, and I can scarcely be accused of having in the instances given omitted such as according to current notions might seem to have the presumption of heredity on their side; on the other hand, I will not omit to point out that, for an incomparably preponderating number of cases, there is wanting all reasonable indication of direct occasion for contagion, which appears also from no mention

being made of the same, as it must be supposed that I would have produced whatever evidence I could produce in that direction. If, however, we now take a review of what has been adduced, we shall find that such direct indications are not totally wanting, and I will specially point out the two importations from non-leprous districts; further, it is shown by instances that certain conditions in the occurrence of leprosy receive their best explanation from contagion, and thereby also some of those cases which otherwise are usually considered without further reflexion as hereditary, become subject to a criticism from which they have hitherto been exempt. I think that I have hereby given at least a beginning for a real indication of the much disputed foci of leprosy, and of the occurrence of leprosy also in families being possibly independent of relationship. If we now compare herewith what we found in Bergen, it appears to me that the probability for contagion rises little by little, while the probability in favour of heredity sinks in a corresponding degree.

As, however, nothing yet has been produced which could be strictly called a proof, we must seek after other things which possibly might confirm or controvert what has hitherto been adduced, and I shall therefore endeavour to extract from the statistics of leprosy in its new form what may be serviceable for this purpose.

These statistics, for the compilation of which we are indebted to Mr. Inspector Hartwig's detailed lists of all the lepers in the country, made voluntarily on his own account by reason of the interest which he has taken in the matter, while the tedious elaboration of the same is also his work, are based on the assumption that all the lepers, so far as is practicable, are registered in that year wherein their disease is supposed to have originated. As nearly all the indications of the duration of the disease are based on the patient's own statement, and this statement, as we have seen, is by no means trustworthy, there will, of course, be many errors in respect of the patients being registered, most probably, years later than the time when their disease really began. For the sake of comparison between the single years, and especially between the periods of five years, this error will probably not be of any great importance, as it will be most likely tolerably equally distributed. The error affects the essential part of the tables, namely, the number of new lepers received yearly; but if one endeavours to correct it by taking a corresponding number of new arrivals for each quinquennium, and carrying it back to the previous quinquennium, the chief results will be the same, only the diminution of new cases which appears everywhere will be more manifest. After many attempts to combine the statements in various manners, I have fixed on the following as the most comprehensive. The districts are

arranged with regard to the asylums to which the largest number of patients are admitted. These asylums are Reitgjärdet near Trondheim, Reknás near Molor, St. Jorgen; the Asylum No. 1, and Lungegaard's hospital near Bergen.

Provinces of Tromsö and Throndhjem Reitgjærdet.

Series of years.	New cases.	Died.	Died in percentage of all lepers.	Placed in asylums.	Placed in asylums in percentage of all lepers.	Remaining.	Year.
1851—1855	304	...	...	...	...	722	1856
1856—1860	348	261	26	23	2·3	700	1860
1861—1865	349	177	17	293	28·0	559	1865
1866—1870	290	155	18	186	21·9	484	1870

Nodre and Sondre Nordmore, Surendal and Sundal Reitgjærdet and Reknæs.

1851—1855	54	...	...	...	...	107	1856
1856—1860	80	32	18	14	8·2	121	1860
1861—1865	86	43	20	45	21·2	117	1865
1866—1870	82	40	24	43	21·5	110	1870

Indre- and Yttre- Romsdal, Ostre-, Nordre-, Vestre-, Indre- Sondmore Reknæs.

1851—1855	87	...	...	...	...	175	1856
1856—1860	101	33	13	28	10·9	190	1860
1861—1865	72	34	13	94	35·9	130	1865
1866—1870	61	35	13	50	26·1	102	1870

Yttre- and Indre- Nordfjord and Nordfjordeidet, Reknæs and Bergen Asylums.

1851—1855	43	...	...	...	...	100	1856
1856—1860	53	19	13	47	33·3	74	1860
1861—1865	42	17	14	19	16·3	78	1865
1866—1870	32	24	22	19	17·2	65	1870

Kinn, Yttre- and Indre- Sondfjord Bergen Asylums and Reknæs.

1851—1855	183	...	...	...	...	433	1856
1856—1860	209	83	13	211	35·0	305	1860
1861—1865	153	61	13	144	31·4	246	1865
1866—1870	112	52	14	137	38·2	168	1870

Yttre-, Midtre-, Indre-Sogn and Lærdal Bergen Asylum.

Series of years.	New cases.	Dicd.	Died in percentage of all lepers.	Placed in asylums.	Placed in asylums in percentage of all lepers.	Remaining.	Year.
1851—1855	128	...	...	...	...	319	1856
1856—1860	83	58	15	117	30·5	205	1860
1861—1865	61	48	18	45	16·9	161	1865
1866—1870	47	30	14	43	20·6	127	1870

The Town of Bergen.

1851—1855	8	...	...	...	...	23	1856
1856—1860	12	1	3	4	11	9	1860
1861—1865	11	1	5	6	30	12	1865
1866—1870	5	2	11	11	64	15	1870

Sondre Bergenhus District.—Bergen Asylums.

1851—1855	195	...	...	...	...	467	1856
1856—1860	173	113	18·5	115	19·0	366	1860
1861—1865	143	98	19·0	71	14·0	337	1865
1866—1870	124	97	21·0	80	17·3	281	1870

Stavanger, Lister, and Mandals Districts.—Lungegaard's Hospital.

1851—1855	77	...	...	...	...	231	1856
1856—1860	66	58	20·0	30	10·6	194	1860
1861—1865	69	55	21·0	16	6·0	189	1865
1866—1870	35	49	21·5	11	5·0	157	1870

Nedenæs, Buskerud, Kristians, Hedemarkens, and Akershus Districts.

1851—1855	22	...	...	...	...	51	1856
1856—1860	6	11	19·0	...	...	44	1860
1861—1865	12	16	28·5	3	6·5	36	1865
1866—1870	9	16	35·5	4	9·0	24	1870

The quinquennium 1851 to 1855, in which only the old St. Jurgens Hospital, Reknæs and Lungegaard's hospital were in operation, has been included, with regard to its new cases; as we may suppose that the statements from later years, after the beginning of the regular counting in 1856, comprise approximately all

the new cases observed in those years. For former years we have not so trustworthy returns as to be able to judge with certainty whether leprosy was on the increase before 1850 or not. In 1836 the number is said to have been 659; in 1845, 1122; and in 1856 the number was, according to what we now know, about 2800. There appears, therefore, to have been a very strong increase; but if we consider how many cases are even now, when the counting is executed by the medical men, overlooked or unknown from one year to another, the number of cases overlooked when regular countings were not instituted, and the countings such as they were depended on the work of non-medical people, must have been still greater. However, I must with Bidenkap assume that the number cannot have been so great as the difference between the figures stated, and that therefore an increase in the number of lepers has really taken place in the present century. This agrees also with the tradition found in many places, and which I have mentioned above. What we may assert with greater confidence is that the numbers in the two quinquenniums 1851—55, and 1856—60 have remained nearly unaltered. The number of new cases, at least, were in these two periods about the same, namely, respectively 1101 and 1131 for the whole kingdom, or about 220 new cases yearly; but in the period 1861—65, 998, or about 190 yearly; and in 1866—70, 797, or about 160 yearly. Since 1860 there is therefore a decrease in the number of new cases, which appears to be steady and not a chance fluctuation. The total number has also decreased in corresponding proportion; in this table there are only noted as remaining those who are at home in the districts, in order to show in what degree the districts are emptied by the receptions into the asylums. As now the number of those remaining is influenced by removal, as also by immigration and erasure from the lists, these numbers are not so trustworthy to guide us as those of the new cases. It should be remarked that the number of those remaining at the end of 1855 is not known, for which reason it was necessary to give it at the end of 1856, and this makes some confusion in the quinquennial periods, but it could not be avoided

It must be our task, if possible, to find the cause of this decrease, and specially to call attention to the new cases. In looking at the tables, the groups of which are formed with reference to the respective asylums, we shall find that these stand very differently in the different districts. In the provincial districts of Sogn, Sondre, Bergenhus and Stavanger, the number of new cases was greater in the first quinquennium than in any of those following; Sogn especially takes a remarkable place in this respect. If we compare the decrease afterwards observable with the number of those placed in the asylums, it does not seem to have had much influence on the decrease of new cases which was already in progress. Neither is the

number of deceases in these dictricts much greater than in others; only in Söndfjord is it in any considerable degree less.

In Nordmöre the number of new cases has remained at about the same point, and is in all the last three periods of five years even considerably larger than in the first, notwithstanding that the admissions to the institutions, have been great in the two last quinquenniums. If the admission into the asylums had had any influence on the number of new cases, we might have expected a decrease in the last quinquennium, as the greatest number of admissions began in the last but one. But, as may be seen from the table, the receptions from this district have not been sufficient to empty the district in any great degree.

The same applies to Romsdal, Söndmöre, and Nordfjord; the diminution in the number of new cases seems here not to have taken place until they began to be received into the asylums, and this diminution is about in the same proportion as elsewhere, but the numbers are so small that scarcely any inference can be drawn therefrom.

This seems, on the contrary, to be the case at Söndfjord. Here is increase in the number of new cases from the first to the second quinquennium; in this second period 35 per cent. of the lepers of the district were placed in the asylums, and in the third quinquennium the number of new cases is diminished by 46; in this period 31·4 per cent. were placed in asylums, and in the fourth quinquennium the new cases were fewer by 41. In no district has the evacuation been so complete as here since the asylums began to receive patients, and till 1870, or in fifteen years, the number of lepers has diminished in the home district from 433 to 168.

In the provinces of Tromsö and Throndhjein the admissions to the institutions seem to have been of importance. The number of new cases increases from the first to the second quinquennium; in the latter period there were placed in the asylums only 2·3 per cent. of the lepers, and in the third period the number of new cases is the same as in the second. But in the third quinquennium there were placed 293 lepers, or 28 per cent., and in the fourth there are 59 fewer new cases.

In order to get larger numbers we may arrange the districts in two large groups, namely, those from which the reception in the asylums began on a larger scale in 1856, and those from which the reception did not begin until 1860. We thus get the following comparison with respect to the number of new cases in the same four quinquenniums:

New cases in—	1851-55.	1856-60.	1861-65.	1861-70.
Province of Bergen and district of Stavanger	634	−38 596	−117 479	−124 355
Other parts of the country	467	533 −68	519 −16	442 −77

Number in the district was—	1856.	1860.	1865.	1870.
Province of Bergen and district of Stavanger	1573	1153	1023	915
Other parts of the country	1055	1055	842	720

Admitted to the asylums:	1856-60.	1861-65.	1866-70.
Province of Bergen and district of Stavanger	524 (24 p. ct.)	301 (18 p. ct.)	301 (22 p. ct.)
Other parts of the country	65 (4 p. ct.)	335 (21 p. ct.)	283 (22 p. ct.)

It is difficult to doubt, according to these figures, that the evacuation in the districts by reception into the asylums has an influence in diminishing the number of new cases; although it may be assumed also, especially in regard to single districts, that this is not the only factor. But before we can pass a final judgment in the matter we must await the experience of future years.

But, if we now assume that the reception into the asylums or isolation has contributed to the decrease of leprosy, it will be of interest to discuss why isolation should operate in that direction. Whether the disease is hereditary or contagious, isolation must be useful; for even if we assume an hereditary abnormity, which may become leprosy without leprous ancestors, yet those who are manifestly leprous must be supposed to transmit the abnormity in a stronger degree than those who are only about to acquire it.

It must therefore be expedient to prevent as great a number of manifest lepers as possible from propagating. The question then arises whether it is possible that the isolation during the short time of fifteen years should have been able to exercise influence on the number of hereditary cases. This seems to be a question which may without any further commentary be answered negatively, especially as the good effects of isolation appear already in the quinquennium immediately following. Such a state of the case is, however, quite consistent with contagion; and if we take up the official statistics we shall find that the decrease does not begin in the year after the commencement of isolation, and not until four or five years afterwards, and this corresponds with the assumption that the patients usually date the commencement of the disease too late. I shall endeavour to elucidate this by an imaginary instance. I suppose that the number of lepers in a district has been for a long series of years uniformly the same, for instance 100, and that there have been constantly 10 new cases yearly and 10 deaths. Of the 100 alive every year, 10 patients infect 10 new ones, and the latter do not become aware of the dis-

ease until five years later. If we set this in the form of a table we have—

For	100	lepers, for instance, in	1851—10	new cases in	1856
„	100	„ „	1852—10	„	1857
„	100	„ „	1853—10	„	1858
„	100	„ „	1854—10	„	1859
„	100	„ „	1856—10	„	1860
			50		

Now, we begin to isolate in 1856, so that I have still in 1856—

For	100	lepers in	1856—10	new cases in	1861
But	90	„	1857— 9	„	1862
„	80	„	1858— 8	„	1863
„	70	„	1859— 7	„	1864
„	60	„	1860— 6	„	1865
			40		

By our isolation we cannot effect any diminution in the number of new cases in 1856—60, because the new cases in these years are really attributable to the previous quinquennium, but the effect does not come to light until the following period, 1861—65. The instance is not quite imaginary. We can suppose the number of lepers existing at the end of 1856, with a proportional addition for the different districts, to represent with tolerable accuracy the average number of the lepers living in each district in the period 1851—55, and likewise the number living at the end of each following quinquennium, with a proportional addition, to represent the average number of the quinquennium; and we find then the number of new cases in each period in several districts standing in a tolerably steady proportion to the number of lepers living at the end of the previous period; and the proportion to the average number will be approximately the same. We have thus in the provinces of Tromsi and Throndjeim—

The number at the end of		1856=722	and 348 new cases in		1856-60
„	„	1860=700	„ 349	„	1861-65
„	„	1865=559	„ 290	„	1866-70

add for Söndfjord—

The number at the end of		1856=433	„ 209	„	1856-60
„	„	1860=305	„ 153	„	1861-65
„	„	1865=246	„ 112	„	1866-70

and for the whole kingdom—

The number at the end of		1856=2628	„ 1131	„	1856-60
„	„	1860=2208	„ 998	„	1861-65
„	„	1865=1865	„ 797	„	1866-70

As, now the average number will be higher than that noted at the end of the quinquennium, the proportion will be that there

will eome about 8 per eent. new eases annually from the lepers living annually in the previous quinquennium. In my instanee I took for the sake of eonvenienee 10 per eent. In some districts there are deviations from this proportion; in some it is over and in others under 8 per eent., but the larger the numbers we work with the nearer we eome to this pereentage. It may, of eourse, here also be objeeted that the time for observation is too short, and I do not wish from these three periods of five years to argue for any regular law; but it seems to me not unreasonable from the number of new eases in 1851—55, whieh is almost aeeurately the same as in 1856—60, to eonelude that the total number of lepers in the kingdom in the period 1846—50 was about the same as in the subsequent quinquennium. The sudden deerease from 1861 would then be of so much more importanee; and I for my part am very much inelined to make the prognosis of the future eourse of leprosy the subjeet of a simple arithmetieal ealeulation; time must show whether sueh a determination would be preeipitate or not.

Although there seem to be rather positive indieations that isolation has eontributed essentially to the undoubted deerease of the new eases, and although sueh an effeet of isolation eannot possibly be made to support the notion of heredity, but must, on the other hand, furnish a strong argument for eontagion, I will not omit to repeat that, by reason of the apparent exeeptions observable, for whieh I ean find no satisfaetory explanation, I will not attempt to pass any deeisive judgment in this question. I shall only insist on one thing. If leprosy is eontagious it would be more probable that the tubereular form, with its more abundant produetions and more frequent uleeration, should be more dangerous than the smooth form; and, therefore, in order that the isolation should work most favorably, the tubereular patients should be speeially isolated, and preferably as early as possible. The first part of this eondition is fulfilled by a preponderatingly great number of tubereular patients being admitted into the asylums. Of those plaeed in the asylum No. 1 there are no fewer than 631 tubereular against 193 of the smooth form.

The seeond part of the eondition is not so well fulfilled; most of the patients not being admitted until several, some times many, years after the evident breaking out of the disease. But their home is at least generally very well evaeuated. Of those patients reeeived into the asylum there are only 62 leprous children known, and of these no less than 57 are reeeived into the asylum.

We will now assume that the asylums have been without any influenee on the diminution, and endeavour to diseover whether any reasonable cause for the same ean be found outside of them. Just as there might be persons of opinion that the eause of the deseendants of the lepers or leprous raees who immigrated to Bergen not

being leprous is that the hereditary disposition is modified by altered conditions of life, so there might also exist the opinion that circumstances during the last fifteen to twenty years round about in the country districts have been so much improved as to counteract the hereditary disposition. Against this view there is first the circumstance that the proportion of family cases seems to be still the same as before, while they ought to be proportionally more numerous. For if the conditions of life should be able to counteract the hereditary disposition, then they must also counteract the occurrence of the so-called spontaneous cases, and as regards these must have incomparably better chance for victory than in combating an hereditary disposition. In the next place, I think that it would be very difficult to point out any essential change in the conditions of life until, perhaps, after 1870, at least in the provinces of Bergen, Romsdal, and Nordmore. How it may be in other districts I have no definite idea. The manner and conditions of life are never in the same place changed all at once, while circumstances demand a slow development; now, there is certainly in the west country a manifest development and improvement of all conditions, but it is, according both to my own observation and to the unanimous testimony of well-informed people, very slow. And according to experience, a long time is required—many generations—for an hereditary peculiarity to disappear. From whatever side we consider the case, it appears that heredity can have no influence on the decrease of leprosy, which may be most reasonably accounted for by its not existing at all.

I have now produced what, according to the extent of my investigations, I have been able to collect in the way of information as to the occurrence of leprosy here in this country, and its conditions, and I have endeavoured to point out those things which appear to me to be influential for the guidance of our judgment. Even if I have not been able to furnish any decisive proof in any direction, I think that I have pointed out a number of phenomena in the occurrence of the disease which find a natural explanation by supposing contagion, but which, on the contrary, must remain unexplained under the supposition of heredity. Leprosy will thus, according to my conception, come into the category of specific diseases which are contagious, but, like specific diseases in general, are not transmitted by inheritance.

But if leprosy is a specific and contagious disease we might, perhaps, expect to find that, like syphilis, it is also transmissible to the offspring. That such is the case can neither be denied nor affirmed. There are some observations of leprosy in so early an age, in the first and second year of children born of leprous parents, that they might be supposed, by reason of the slow development of this disease, to have got the disease while in the uterus. But the cases

are so extremely rare, and the leprous parents who have children are so many, that it is not probable. Most of the children who have leprosy are over five years of age, and it seems, indeed, reasonable to suppose that they have contracted the disease after birth. What might be most likely to direct our thoughts to a transmission of the disease to the ova is the fact that the testicles, according to my later investigations, are always leprous, and, as it appears, from the very beginning of the disease. In a patient who took service with a leprous master and became leprous the year after, one year later was placed in the Lungegaard Hospital, and died there half a year afterwards, it was found that, besides the skin, the liver, the spleen, the nerves and the testicles were leprous, and I was able to demonstrate this to Dr. V. Carter, from Bombay, who was just then residing in Bergen, to study our arrangements with respect to leprosy. Now, the products of the leprous affection in the testicles are found not only in the intertubular connecting tissue, but also in the seminal canals. As to this point I was for some time in doubt, but I have now obtained preparations in which it appears quite unmistakably; for the retrograde elements may lie in rows in the seminal canals, and by their magnitude partially enlarge them, so that a canal isolated for any considerable length takes the appearance of a string of small beads. I have not yet found any corresponding affection of the ovaries. It is now sufficiently ascertained that leprous men can procreate children; and when leprosy attacks the testicles in the manner mentioned, it may easily be supposed possible that the leprous contagion may go together with the spermatozoa. This anatomical discovery in leprous testicles gave me the idea that the syphilitic orchitis might perhaps have some influence on the transmission of syphilis to the offspring. With reference to leprosy nothing certain can be said, as we have seen.

The theory of leprosy being a contagious disease, and not hereditary, has been brought forward, in 1869, by Boynat-Landré, in his book 'La Contagion seule cause de la Lepre.' However, we differ in our views respecting heredity. While Landré quotes the discrepancy between the transmission to offspring of syphilis and the heredity claimed for leprosy as an argument against the latter, this discrepancy, in my opinion, might just be adduced as strengthening the notion of the heredity of leprosy. On the other hand, there are parts in Landré's book the importance of which can scarcely be shaken, and which have hitherto met with no opposition namely, the history of the course of leprosy in Surinam, and the cases related of leprosy in descendants from European parents. Landré's representation stands to me in these respects as the most convincing evidence hitherto given of the contagious nature of leprosy.

While leprosy may be thus indirectly proved to be a specific

disease by demonstrating its contagiousness, it would, of course, be the best if a direct proof could be given. I will briefly mention what seems to indicate, that such proof is, perhaps, attainable. There are to be found in every leprous tubercle extirpated from a living individual—and I have examined a great number of them—small staff-like bodies, much resembling bacteria, lying within the cells; not in all, but in many of them. Though unable to discover any difference between these bodies and true bacteria, I will not venture to declare them to be actually identical. Further, while it seems evident that these low forms of organic life engender some of the most acute infectious diseases, the attributing of the origin of such a chronic disease as leprosy to the apparently same matter must, of course, be attended with still greater doubts. It is worthy of notice, however, that the large brown elements found in all leprous proliferations in advanced stages, of which I have in 1869 already given engravings, republished in 'Leprous Diseases of the Eye,' by O. B. Bull and G. A. Hansen, bear a striking likeness to bacteria in certain states of development, as these are represented by Klebs in the first number of 'Zeitschrift für Experimentelle Pathologie und Pharmacologie;' and further, that in almost every preparation from a leprous tubercle, made with the utmost care to avoid contamination and kept for a number of days in the damp chamber, are developed conglobate masses of spherical bacteria or zooglœa. It would be desirable that other inquirers should direct their researches to this point.

I could further point out several features in the pathology of leprosy that tell strongly in proof of its specific nature, but I must desist from doing so in this article. Perhaps I may make it the subject of a further communication.


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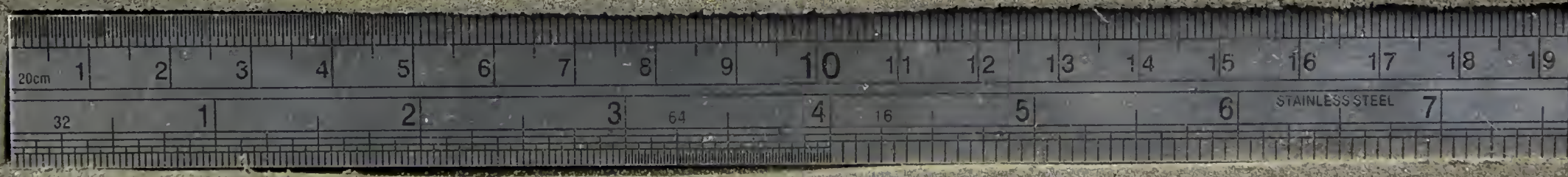
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